Purulent pericarditis

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Case Report

A 77 year old Caucasian male was found unresponsive at his residence. He had no significant medical history, except for severe anemia treated with a transfusion two months prior.

At autopsy, external examination of the body revealed a well-developed, well-nourished, thin male with no remarkable features. Internal examination of body cavities revealed cloudy yellow fluid within each pleural cavity. The pleural surfaces were focally purulent, particularly inferomedially. The pericardial cavity was completely replaced with thick purulent fluid adherent to both the epicardium and pericardium (Fig. 1-4). Further cardiovascular exam revealed cardiomegaly (heart weight 520 g) with focal areas of mild to severe atherosclerosis in the coronary artery system. There was concentric left ventricular hypertrophy, measuring up to 1.7 cm, along with marked biventricular dilatation. The endocardium was unremarkable. Apart from aortic atherosclerosis and pulmonary emphysema, all other organ systems were grossly unremarkable.

Microscopic examination of the heart revealed fibrinous and purulent pericarditis. The lungs showed emphysema, purulent pleuritis, and focal pneumonia. Both pericardial fluid and blood cultures grew *Streptococcus pneumoniae*. The urine drug screen was positive for only Caffeine and Chlorpheniramine, and a serum drug screen was positive for ethanol at 8 mg/dL.

Based on the autopsy findings, the cause of death was determine to be purulent pericarditis with associated pneumonia and presumed sepsis, with underlying hypertensive and atherosclerotic cardiovascular disease and pulmonary emphysema.

Discussion

The most likely source of the pericardial infection in the case described is infection due to untreated pneumococcal pneumonia. Pneumococcal pneumonia is the most common clinical presentation of pneumococcal disease among adults. Its complications include empyema and endobronchial obstruction with atelectasis and lung abscess formation [1]. Purulent pericarditis, while quite rare today, is another well-demonstrated complication of untreated pneumococcal pneumonia and sepsis [2]. It may occur by direct spread of an intrathoracic, myocardial, or subdiaphragmatic focus, a perforating injury to the chest wall, or hematogenous dissemination [3,4]. In this case, it is difficult to say with certainty whether there was direct spread due to bacteria migrating directly from the lung tissue itself, or if there was a subsequent bacteremia first and a following invasion of the pericardial sac [1].

Prior to the advent of penicillin in 1943, pneumococcal pericarditis was the most common cause of purulent pericarditis. The disease was also seen most often in children [1,5]. Since 1943, the widespread use of both antibiotics and the pneumococcal vaccine are responsible for a decline in the overall incidence of purulent pericarditis by a factor of three [2]. The median age at diagnosis has also increased from 21 years before penicillin use to 49 years old thereafter [3]. Now the disease is seen more commonly in older patients with underlying pericardial disease, immunosuppression, malignancy or following surgery. Only 20% of cases seen today are a result of spread from pleuropulmonary disease

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[6]. Instead, the incidence of both gram negative and anaerobic organisms causing purulent pericarditis has, unsurprisingly, increased. Candida albicans is another causative organism on the rise, seen most commonly in immunocompromised patients [3]. No differences have been found in the clinical features of cases due to anaerobic bacteria versus those due to aerobic and facultative bacteria [4]. There is some concern that an increasing number of pneumococci are highly penicillin resistant (MIC > 1.0 mg/mL) and that this may cause a reemergence of pneumococcal pericarditis [6,4]. Purulent pericarditis, therefore, remains an important topic of discussion.

While the incidence of purulent pericarditis has decreased, mortality remains high. Nearly 100% of unrecognized cases are fatal, and over 50% of cases, as above, are diagnosed at autopsy [3]. The low ante-mortem diagnosis rate is evidence of the diagnostic challenge presented by purulent pericarditis. A major reason for this is the extremely variable presentation. Typical pericarditis features including pulsus paradoxus, right heart failure, chest pain, or a pericardial friction rub are frequently absent [5,7]. Diagnosis is often delayed further if a focus of infection has previously been identified due to a tendency to attribute the more reliable, but less specific symptoms, like tachycardia or pyrexia, to the underlying infection itself [2,3]. A high clinical suspicion is, therefore, paramount to timely diagnosis and treatment.

Echocardiography is the investigation of choice if purulent pericarditis is suspected [8]. As a non-invasive tool that both identifies pericardial effusions and acts as a guide for pericardiocentesis, echocardiography has greatly improved diagnosis [2]. Transthoracic echocardiography can demonstrate pericardial fluid in virtually all patients with bacterial pericarditis; however, it cannot distinguish between purulent fluid collections and sterile or inflammatory effusions. The ultimate diagnosis depends on the pathologic analysis of the fluid from pericardiocentesis [4]. Pericardial fluid should be sent for extensive microbiologic testing including gram stain, acid-fast bacilli stain, fungal stain, culture for aerobic, anaerobic, and fungal pathogens and a complete cell count with differential. Fluid analysis in purulent pericarditis may also demonstrate polymorphonuclear leukocytosis, low glucose, high protein, and elevated lactate dehydrogenase. The presence of frank pus, as seen in the reported case, is 100% sensitive [3]. X-ray findings often include pulmonary infiltrates, pleural effusions, and mediastinal widening [3]. EKG may also be used for diagnosis. Generalized ST segment elevation without reciprocal changes are the most common changes seen in pericarditis; however, EKG may be normal in over 35% of cases [2,3].

The potential for a successful outcome in purulent pericarditis is dependent upon early diagnosis, which allows urgent and aggressive treatment. Pericardiocentesis is not only diagnostic, but may be life-saving, as well. Urgent pericardial drainage is necessary in purulent pericarditis. High dose, empiric, intravenous antibiotics should also be started immediately. A more tailored antibacterial therapy can then be used according to the results of drug sensitivity testing from pericardial fluid and blood cultures. IV therapy should be continued until fever and clinical signs of infection have resolved and the white blood cell count has returned to normal. Even with prolonged therapy, however, cardiac tamponade and death is not unusual [4]. With therapy, mortality rates are near 40%. While still high, this is a drastic decrease in comparison to untreated cases, in which mortality rates climb to almost 100% [3].

Purulent pericarditis is a severe disease with a fulminant course that has become less common since the advent of antibiotics. It remains a life-threatening complication of infective illness, especially pneumonia, making it an important topic for discussion. Its signs and symptoms are often extremely

varied and can be masked by the already known focus of disease, which has led to a very high post-mortem diagnosis rate. Untreated cases of purulent pericarditis are almost uniformly fatal, while treated cases have a mortality rate near 40% [3]. While still high, the drastic increase in survival that is seen with timely diagnosis highlights the importance of increased clinical awareness of this condition.

References

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